testing vaccination strategies. One of the main benefits is that for parameterization and identification no abstract values are used, so objectivity and traceability are assured.

PMC33

CRITICAL REVIEW OF ECONOMIC MODELS IN TYPE 2 DIABETES

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OBJECTIVES: To identify and critically appraise cost-effectiveness models developed to evaluate type 2 diabetes (T2D) treatments and to assess which types of treatment effects they capture. **METHODS:** A systematic search was performed in MEDLINE, EMBASE, Centre for Reviews and Dissemination databases at the University of York, and Health Economic Evaluation Database for the period to September 2008. The websites of Health Technology Assessment (HTA) bodies in different countries were also screened for relevant models. For each of the identified original models, details of the structure, data in- and outputs and consistency were extracted and critically appraised using published criteria. **RESULTS:** 78 articles and 41 HTAs reporting relevant economic evaluations were identified. There were ten models with multiple publications, and a further ten models with one associated publication. The critical review demonstrated that most of the existing models had the same fundamental structure, used similar microsimulation techniques and were based on the same key data sources. However, the process for identification of relevant data and their synthesis, as well as the selection of outcomes was, at times, inconsistent and lacked transparency. The models differed according to which diabetes complications and treatment-related adverse events were captured. For example, just one model incorporated changes in patient weight, despite the fact that weight gain can be a side effect of some treatments, and weight loss a potential benefit of others. **CONCLUSIONS:** Whilst many economic models exist in T2D, most share common features such as the model type. Identified shortcomings are lack of transparency in data identification and evidence synthesis as well as the selection of the modelled outcomes. Future models should aim to include all relevant treatment outcomes, whether these relate to effects on underlying diabetes and its complications or to short- or long-term side effects of treatment.

PMC34

SEROTYPE-SPECIFIC TRANSMISSION DYNAMICS OF INVASIVE PNEUMOCOCCAL DISEASE AFTER VACCINATION WITH 7-VALENT PNEUMOCOCCAL CONJUGATE VACCINE

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OBJECTIVES: After the introduction of the 7-valent pneumococcal conjugate vaccine (Prevnar®) in the US, the incidence of invasive pneumococcal disease (IPD) caused by the 7 vaccine serotypes declined dramatically in vaccinated children as well as unvaccinated adults (via herd effect benefit). In 2008, a transmission dynamic equation model was developed to capture direct and indirect vaccination effects. This model accurately predicted the total incidence of IPD (caused by all serotypes) after Prevnar® introduction. This original model was refined in the present analysis to predict the dynamics of IPD caused by specific serotypes. **METHODS:** The model simulates the acquisition of asymptomatic carriage of pneumococci and the development of fatal and non-fatal IPD among vaccinated and unvaccinated individuals aged <2, 2–4, 5–17, 18–49, 50–64, and >= 65 years old. Categories of pneumococcal serotypes include PCV7-type (4, 6B, 9V, 14, 18C, 19F, and 23F) and non-PCV7-type (all others). The model was calibrated by approximating serotype-specific US IPD surveillance data from the years 1998–2006. **RESULTS:** The previous model structure fit the disease incidence caused by the PCV7 serotypes quite well, but was inadequate to predict increases in disease caused by the non-PCV7 serotypes due to serotype replacement. Additionally, the surveillance data showed limited increase in IPD caused by non-PCV7 serotypes in the vaccinated <2-year-old group. A subsequent recalibration and reformulation resulted in a revised model able to replicate closely the observed IPD incidence stratified by pneumococcal serotypes. **CONCLUSIONS:** The revised model validates the accuracy of the original model to replicate the incidence of IPD caused by PCV7 serotypes and may be used to predict the future incidence of disease given the increases in IPD caused by non-PCV7 serotypes.

PMC35

SCHIZOPHRENIA MODELING: MARKOV MODEL WITH MONTE-CARLO MICROSIMULATION

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Pharmacological strategies for schizophrenia have received increasing attention due to the development of new and costly drug therapies. Evaluating their relative costs and benefits in Canada requires modeling the natural course of schizophrenia. **OBJECTIVES:** To develop a Markov model with 1st-order Monte-Carlo simulations to simulate the natural course of newly diagnosed schizophrenic patients. **METHODS:** Six discrete disease states defined the Markov model: 1): first episode—FE; 2) low dependency state—LDS; 3) high dependency state—HDS; 4) Stable; 5) Well; and 6) Death. Patients' movements between these disease states defined 17 probability transitions to be estimated. The model was based on data from the *Régie de l'assurance maladie du Québec* and *Med-Echo* databases. All individuals aged 0–60 years with a newly diagnosis of schizophrenia between 1998 to 2006 were first identified by ICD-9 codes. Using this data, 5 Cox proportional hazard models for competing risks were used to estimate the 17 probabilities of transition. Validation was conducted by comparing the model's probability transitions' predictions with the published literature. **RESULTS:** A total of 12,754 individuals were identified as newly diagnosed patients with schizophrenia. After the FE of schizophrenia, 69.8% of patients passed in LDS, 11.2% in HDS, 1% in death state and 18% in Well state. The mean transition probabilities after one year of follow-up were: FE to Well at 0.28 (±SD = 0.10), FE to HDS at 0.11 (±0.05), FE to LDS at 0.60 (±0.08) and respectively FE to Death at 0.01 (±0.01). The corresponding values were similar to those obtained from other published models. **CONCLUSIONS:** This model is the first Canadian model incorporating transition probabilities adjusted for individual risk factors profiles using Canadian data. Future applications will include pricing and cost-effectiveness of new therapies for schizophrenia.

PMC36

CREATING NATIONAL WEIGHTS FOR PRIVATE INSURANCE DATABASE

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OBJECTIVES: To create national weights to project a private database to the US insured population controlling not only for socio-demographic factors but also health status. **METHODS:** The Medicare Expenditure Survey was used as the basis of adjustment methodology. First, we subset the data source to the study population, then used multivariate logistic regression to construct demographics and case-mix based weights that were applied to make the data similar to the national sample. Propensity Score Matching and Raking algorithm is combined to create the adjusted weights. The socioeconomic characteristics included in the model were the age of the head of the household, percentage of the patients who were female, race, geographic region and income level. We derived two variables to capture general health status of the member. First, Charlson Index scores were generated to capture the level and burden of comorbidity. Secondly, we created an indicator variable to represent patients with chronic conditions. This variable was derived by convening two physician panels to review all medical conditions reported by the survey sample. **RESULTS:** Private data were more likely to be male, white, older, and chronic ($p = 0.0000$). Adjusted weight values for the Commercial group ranged from 13.47 to 26.39 with median 16.35. The projected US population by private database and MEPS data were similar in terms of socioeconomic and clinical categories. As an outcomes measure, the predicted annual statin users from private data was 6,973,034. Statin users are predicted as 6,709,438 using MEPS data with MEPS weights. **CONCLUSIONS:** National projection of a private database requires adjustment from not only demographic factors but also case-mix differences related to health status. The created weights successfully balanced the population in terms of co-morbid conditions and chronic conditions as well as demographic factors.

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COMPARISON OF RISK ADJUSTMENT MODELS IN OUTCOMES RESEARCH

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OBJECTIVES: Matching and regression analysis are two approaches to estimate average treatment effect. Different matching techniques provide different results. Moreover, matching cannot control for unobserved bias. Using ProbChoice algorithm and Rosenbaum bounding approach, we aim to show how to choose strongly unmeasured variable must influence the selection process to undermine the implication of matching and regression analysis. **METHODS:** The Surveillance, Epidemiology, and End Results (SEER) Data is used for the analysis. For each patient, their hospital of care and associated hospital volume is computed. Patients in the high and low volume hospitals are matched in seven different ways in terms of demographic and clinical characteristics. Treatment costs are compared. The best technique is chosen by ProbChoice algorithm. Rosenbaum bonds estimated and Mantel and Haenszel test statistics is calculated to provide evidence on the degree to which any significance results hinge on unconfoundedness assumption. **RESULTS:** A volume cohort was constructed consisting of 19,375 female SEER-Medicare patients, aged 65+, suffering an in situ and/or invasive breast cancer during 2003–2005 with surgical treatment performed at 567 hospitals. Mahalobis matching is the one who created the best balanced comparable sets. After the matching, samples were similar in terms of race, comorbidity and adjuvant therapies. Under the assumption of no hidden bias, costs were lower of the high volume hospitals. ($p = 0.000$). Results were insensitive to a bias that would double the odds of being treated high volume hospitals but sensitive to a bias that would triple the odds. **CONCLUSIONS:** There exist several matching techniques and the results depend the type of matching chosen. One needs to chose the technique best suitable for the data. Rosenbaum bonds provides evidence on sensitivity of the estimated results with respect to unobservable factors that is not controlled by propensity score matching.